IN THE CLAIMS:

All claim amendments are made without prejudice or disclaimer. Please amend the claims as follows:

1. (Original) An inhibitory peptide capable of inhibiting β pleated sheet formation in amyloid β-peptide said inhibitory peptide being a βsheet breaker peptide analog designed by chemical modification of a βsheet breaker peptide capable of inhibiting β pleated sheet formation in amyloid β-peptide.

2. (Original) The inhibitory peptide of claim 1 wherein said βsheet breaker peptide is a 5 residue Alzheimer inhibitor peptide iA β5 (Leu-Pro-Phe-Phe-Asp SEQ ID NO:1).

3. (Currently amended) An inhibitory peptide capable of inhibiting conformational changes in prion PrP protein associated with amyloidosis, said inhibitory being a βsheet breaker peptide analog designed by chemical modification of a βsheet breaker peptide capable inhibiting said conformational changes in ~~prior~~ prion PrP protein associated with amyloidosis.

4. (Original) The inhibitory peptide of claim 3 wherein said βsheet breaker peptide is 13 residue prion inhibitor peptide iPrP 13 (Asp Ala Pro Ala Ala Pro Ala Gly Pro Ala Val Pro Val, SEQ ID NO:2).

5. (Original) The inhibitory peptide of claim 4 wherein said chemical modification is achieved by a process selected from the group consisting of: alteration of the N- and C- terminal ends of said prion inhibitor peptide iPrP13; replacing at least one residue of said prion inhibitor peptide iPrP13 with α-aminoisobuiric acid (Aib); methylation of the α carbon of at least one residue of said prion inhibitor peptide iPrP13 with a D-enantiomeric residue, forming head-to-tail cyclization of said prion inhibitor peptide iPrP13, replacing amide bonds in said prion inhibitor peptide 1PrP13 with an amide bond surrogate; and combination thereof.

6. (Original) The inhibitory peptide of claim 5 wherein said alteration of the N- and C-terminal ends of said prion inhibitor peptide iPrP13 is achieved by a process selected from acetylation, amidation, desamination, alcoholization and combinations thereof.

7. (Original) The compound of claim 6 wherein said inhibitory peptide is selected from the group consisting of: ac-Asp Ala Pro Ala Ala Pro Ala Gly Pro Ala Val Pro Val-am, des-Asp Ala Pro Ala Ala Pro Ala Gly Pro Ala Val Pro Val-am, ac-Asp Ala Pro Ala Ala Pro Ala Gly Pro Ala Val Pro Val-alc, and des-Asp Ala Pro Ala Ala Pro Ala Gly Pro Ala Val Pro Val-alc.

8. (Original) The inhibitory peptide of claim 5 wherein said inhibitory peptide is selected from the group consisting of

Asp Ala Alb Ala Ala Aib Ala Gly Aib Ala Val Aib Val (SEQ ID NO: 4);

Asp Ala Pro Ala Ala Pro Ala Gly Pro Ala (Me) Val Pro Val;

Asp Ala Pro Ala Ala Pro Ala Gly Pro Ala Val Pro (Me) Val;

Asp Ala Pro Ala Ala Pro Ala Gly Pro Ala (Me) Val Pro (Me) Val;

asp ala pro ala ala pro ala gly pro ala val pro val;

asp Ala Pro Ala Ala Pro Ala Gly Pro Ala Val Pro val;

asp Ala Pro ala Ala Pro ala Gly Pro ala Val Pro val;

Aspψ[CH_2CH_2]Ala Proψ[CH_2CH_2]Ala Ala Proψ[CH_2CH_2]Ala Gly Proψ[CH_2CH_2]Ala ValProψ[CH_2CH_2]Val;

Aspψ[CH_2S]Ala Proψ[CH_2S]Ala Ala Proψ[CH_2S]Ala Gly Proψ[CH_2S]Ala Val Proψ[CH_2S]Val;

Ac-Asp Ala Proψ[CH_2CH_2]Ala Ala Proψ[CH_2CH_2]Ala Gly Proψ[CH_2CH_2]Ala Val Pro Val-Am.

asp Ala Proψ[CH_2CH_2]Ala Ala Proψ[CH_2CH_2]Ala Gly Proψ[CH_2CH_2]Ala Val Pro val;

Ac-Asp Ala Proψ[CH_2S]Ala Ala Proψ[CH_2S]Ala Gly Proψ[CH_2S]Ala Val Pro Val-Am;

asp Ala Proψ[CH_2S]Ala Ala Proψ{CH_2S]Ala Gly Proψ[CH_2S]Ala Val Pro val;

Ac-Asp Ala Aib Ala Ala Aib Ala Gly Aib Ala Val Pro Val-Am (SEQ ID NO:5);

Ac-Asp Ala Proψ[CH_2CH_2]Ala Ala Proψ[CH_2CH_2]Ala Gly Proψ[CH_2CH_2]Ala Val Pro (Me) Val;

Ac-Asp Ala pro Ala Ala Proψ{CH_2CH_2]Ala Gly pro Ala Val Pro Val-Am;

asp Ala Proψ[CH_2CH_2]Ala Ala Proψ[CH_2CH_2]Ala Gly Proψ[CH_2CH_2]Ala Val Pro (Me) Val;

asp Ala Aib Ala Ala Proψ[CH_2CH_2]Ala Gly pro Ala Val Pro (Me)Val (SEQ ID NO:6);

asp Ala Aib Ala Ala Proψ[CH_2S]Ala Gly pro Ala Val Pro (Me) Val;

asp Ala Proψ{CH_2S]Ala Ala Proψ{CH_2S]Ala Gly Proψ[CH_2S]Ala Val Pro (Me) Val;

Ac-Asp Ala Aib Ala Ala Proψ[CH_2CH_2]Ala Gly Aib Ala Val Pro (Me) Val (SEQ ID NO:7);

Asp Ala pro Ala Ala Proψ[CH_2CH_2] Ala Gly pro Ala Val Pro Val

Asp Al Aib Ala Ala Proψ[CH_2CH_2] Ala Gly Aib Ala (Me) Val Pro Val Pro Val

Ac-Asp Ala Proψ[CH_2S]Ala ala Proψ[CH_2S]Ala gly Proψ[CH_2S]Ala (Me) Val Pro Val-Am;

Ac-Asp Ala Aib ala Ala Proψ[CH_2CH_2]Ala Gly pro Ala Val Pro (Me) Val;

asp Ala Aib Ala Ala Proψ[CH_2CH_2]Ala Gly Aib ala Val Pro Val-Am;

Ac-Asp Ala pro Ala Ala Proψ[CH_2CH_2]Ala gly pro Ala (Me) Val Pro Val-Am;

asp Ala Proψ[CH_2CH_2]Ala Ala Proψ[CH_2CH_2]Ala gly Proψ[CH_2CH_2]Ala val Pro val;

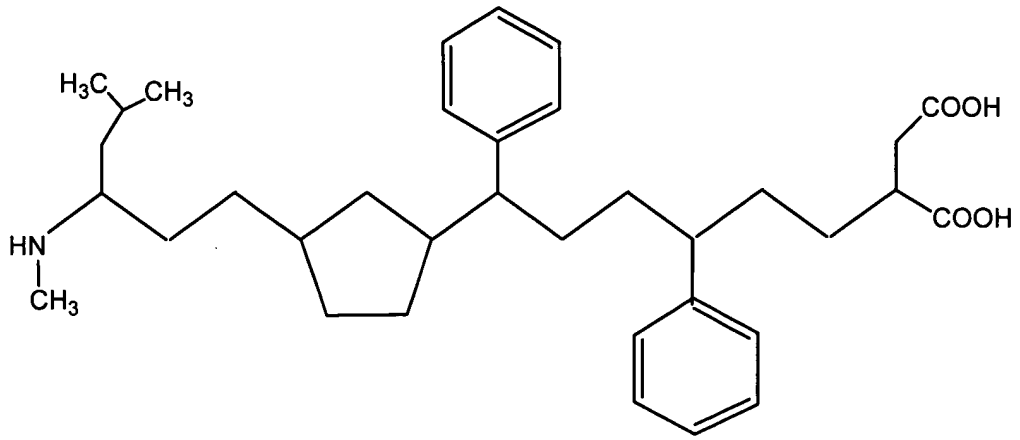
Ac-Asp Ala pro Ala ala Aib Ala gly pro Ala (Me)Val Pro Val-Am (SEQ ID NO:8);

Asp Ala pro Ala Ala Proψ[CH_2CH_2] Ala Gly pro Ala Val Pro Val;

Asp Ala Aib Ala Proψ[CH_2CH_2] Ala Gly Aib Ala (Me) Val Pro Val; and

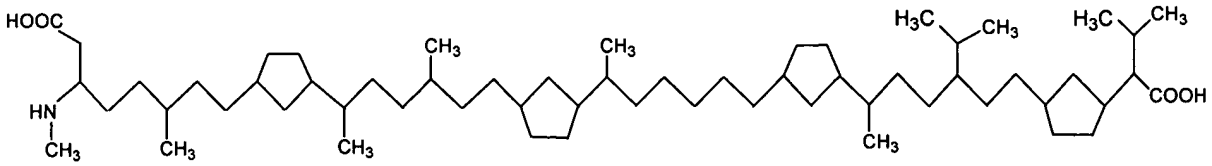
Asp Ala Pro Ala Ala Pro Ala Gly pro Ala Val Pro Val

9. (Original) A peptide mimetic with the following structure:



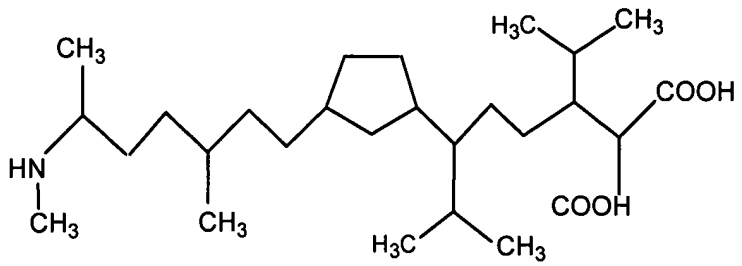
PMiAβ5

10. (Original) A peptide mimetic with the following structure:



PMiPrP13

11. (Original) A peptide mimetic with the following structure:

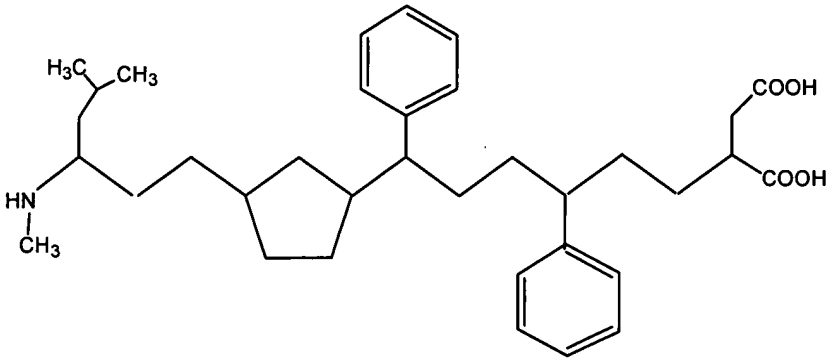


PMiPrP5

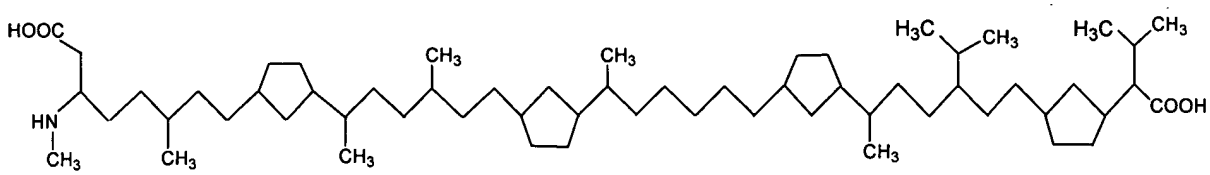
12. (Currently Amended) A method for reducing the formation of amyloid or amyloid like deposits involving abnormal folding into β sheet structure of amyloid β peptide or for reducing the amount of said amyloid β peptide which has already formed into a beta sheet structure comprising bringing into the presence of said amyloid β peptide either prior to or after the abnormal folding thereof into a β sheet structure, an effective amount of a β-sheet breaker peptide analog designed by chemical modification of a β-sheet breaker peptide capable of inhibiting β pleated sheet formation in an amyloid β-peptide~~the peptide of claim 1~~.

13. (Currently amended) A method for reducing the formation of amyloid or amyloid like deposits involving conformational changes in prion PrP protein or reducing the amount of said prion PrP protein which has already formed into amyloid or amyloid-like deposits comprising bringing into the presence of said ~~peon~~ prion PrP protein either prior to or after said conformational changes thereof into amyloid deposits an effective amount of the peptide of claim 3.

14. (Original) A method for reducing the formation of amyloid or amyloid like deposits by administration of a peptide mimetic selected from one of the group consisting of:

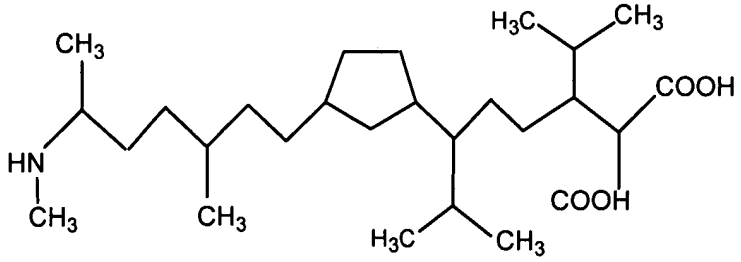


PMiAβ5



PMiPrP13

and



PMiPrP5

15. (New) The method according to claim 12, wherein the β-sheet breaker peptide comprises iAβ5 (SEQ ID NO:1).

16. (New) The method according to claim 12, wherein the inhibitor peptide analog has side-chain groups corresponding to amino acids Leu, Pro, Phe, Phe, and Asp.

17. (New) The method according to claim 12, wherein the chemical modification is achieved by a process selected from the group consisting of: an altered N- or C- terminal end of the peptide; replacement of a residue of the peptide with α-aminoisobutyric acid (Aib); modification of an α carbon of the peptide selected from the group consisting of methylation, alkylation, dehydrogenation, and combinations thereof; amidation; replacement of an L-enantiomeric residue with a D-enantiomeric residue, head-to-tail cyclization of the peptide; replacement of an amide bond in the inhibitor peptide with an amide bond surrogate; and combinations thereof

18. (New) The method according to claim 17, wherein the alteration of the N- or C-terminal end is achieved by a process selected from acetylation, amidation, desamination, alcoholization and combinations thereof.

19. (New) The method according to claim 12, further comprising:

administering an effective amount of a five residue inhibitor peptide analog to a subject, wherein the five residue inhibitor peptide analog has side-chain groups of amino acids Leu, Pro, Phe, Phe, and Asp, wherein the five residue inhibitor peptide analog further comprises a modification selected from the group consisting of: an altered N- or C- terminal end; replacement of a residue with α-aminoisobutyric acid (Aib); methylation of an α carbon; alkylation of an α carbon; dehydrogenation of an α carbon; amidation; replacement of an L-enantiomeric residue with a D-enantiomeric residue, head-to-tail cyclization; replacement of an amide bond with an amide bond surrogate; and combinations thereof; and

reducing the formation or amount of amyloid deposits involving an amyloid β peptide folding into a β sheet structure.

20. (New) The method according to claim 19, wherein the alteration of the N- or C-terminal end is achieved by a process selected from acetylation, amidation, desamination, alcoholization and combinations thereof.

21. (New) The method according to claim 19, wherein at least 81.5% of the five residue inhibitor peptide analog remains uncleaved when incubated *in vitro* with human microsomes at 37 °C for one hour.

22. (New) A method for reducing formation or amount of an amyloid deposit involving an amyloid folding into a β sheet structure, the method comprising:
contacting the amyloid peptide, either prior to or after the abnormal folding thereof into a β sheet structure, with a means for reducing the formation or amount of an amyloid deposit.

23. (New) The method according to claim 22, comprising administering the means for reducing the formation or amount of an amyloid deposit to a subject believed to suffer from a disease selected from the group consisting of Alzheimer's disease, Down's syndrome, Primary systemic amyloidosis, Secondary systemic amyloidosis, Familial Mediterranean fever, Creutzfeldt-Jakob disease, Gerstmann-Strausslet-Scheinker syndrome, Senile systemic amyloidosis, Familial amyloid polyneuropathy, Hemodialysis-related amyloidosis, and Hereditary cerebral amyloid angiopathy.

24. (New) The method according to claim 22, wherein the disease is Alzheimer's disease.

25. (New) A method of detecting a amyloid β based disease in a subject, the method comprising:
administering a five residue peptide analog to a subject, wherein the five residue inhibitor peptide analog has side-chain groups of amino acids Leu, Pro, Phe, Phe, and Asp, wherein the

five residue inhibitor peptide analog further comprises a modification selected from the group consisting of: an altered N- or C- terminal end; replacement of a residue with α-aminoisobutyric acid (Aib); methylation of an α carbon; alkylation of an α carbon; dehydrogenation of an α carbon; amidation; replacement of an L-enantiomeric residue with a D-enantiomeric residue, head-to-tail cyclization; replacement of an amide bond with an amide bond surrogate; and combinations thereof;

binding the five residue peptide analog to an amyloid β peptide in the subject; and

visualizing binding of the five residue peptide analog.

26. (New) The method according to claim 25, wherein visualizing binding comprises visualizing a fibril deposit.